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ORIGINAL ARTICLES.

THE CONSTRUCTION AND USE OF THE OPHTHALMOMETER—AN EXPLANATION AND A CORRECTION.

BY SWAN M. BURNETT, M.D., PH.D., WASHINGTON, D. C.

As the first account, in English, of the construction and use of the ophthalmometer appeared in my Treatise on Astigmatism published in 1887, it seems like going back to somewhat ancient history to bring it up now; and I should not again obtrude it upon a professional public, who have of late been treated to quite an avalanche of papers *pro* and *con* as to the value of the instrument, were it not that that description contains an important error which has not only escaped detection hitherto, but has been perpetuated in other places. So far as I know that is the only description in an English book which gives in any considerable detail the optical principles on which the instrument is constructed, and it is important that those who are studying the apparatus should be correctly informed regarding the arrangements and relations of the parts so as to

have an intelligent conception of the phenomena observed[1].

As the ideas in respect to what the ophthalmometer really does seem to be very vague, not to say incorrect, even among those who are using it regularly in practice, it should be understood, in the first place, that its underlying principle is the measurement of the radius of curvature of the cornea by means of an image reflected from its surface. If you know the size of the object, the size of the reflected image and the distance of the object from the reflecting surface, you have all the data at hand for the determination of the radius of a convexly curved surface. Javal's instrument is simply a convenient means for attaining this end.

As the chief, not to say the sole, use of the instrument is for the detection of corneal astigmatism, what is really to be determined is the difference in the curvature of the two opposite meridians, and this is made manifest to the eye of the observer by a difference in the size of the corneal reflection in these two meridians. The optical law is that the surface or meridian of greatest curvature (shortest radius) shall give the smallest image and that of the least curvature and longest radius the largest image; and the instrument of Javal is so constructed as to enable the observer to measure this and the amount of difference in the two meridians at a glance. This it does by a doubling of the corneal image by means of a prism. When the corneal image is 3 mm. in diameter and the telescope is properly adjusted, the edges of these two images, produced by the prism, are in contact, and, conversely, when they are in contact they must each have a diameter of 3 mm. When the double images are separated the corneal image is smaller than 3 mm., indicating a stronger corneal curvature, and when they overlap it is larger than 3 mm. with a greater corneal curvature. In the instrument the lateral boundaries of the object whose

[1]I find, since writing the above, a full account of the optical principles of the ophthalmometer in the last edition of Dr. F. Valk's "Errors of Refraction." He does not fall into the same error that I did, but he accepts the old and incorrect index of refraction (1.35) for the cornea in constructing his table of Dioptry Reciprocals.

corneal image we measure are two white bands (or mires) one of which is graded in steps, and it is so arranged that the amount of over-lapping, when there is any, can be read off on these steps, each one of which represents a dioptry of refraction. And it is just here that the error into which I have fallen comes in.[2] On pages 130 and 131 of my treatise I say: "Moreover the meridian in which there is a crossing of the bands is the less refracting. The fact of the two images overlapping shows that they have a diameter greater than 3 mm., and consequently the surface giving them must have less curvature than that giving them with the edge in contact, and in order to have them thus in contact the object must be made smaller by moving M′ on the arc toward M. If the images of the bands separate in moving the arc from its initial position where they are in contact it shows that the first meridian is the less refracting with a larger radius of curvature." On page 130 in the description of Fig. 38 the same fact is stated, "A, the meridian of greatest curvature, B, meridian of least curvature (longest radius)."

All these statements are correct as regards the instrument as Javal first constructed it, and as it was described by him and also by Gavarret in an article in the *Revue Scientifique*, 15 Juillet, 1882. But when we come to apply these principles to the instrument as now made we find that they do not hold in practice, for we have the *crossing* of the bands in the meridian of *greatest* curvature where the corneal image is the smallest. For instance, the bands being in contact in the horizontal meridian, if the arc is turned to the vertical meridian and there is a crossing of two steps, that signifies that we have an astigmatism of 2D, according to the rule, in which the vertical meridian of the cornea is the most strongly curved and most highly refracting. This seems in direct opposition to the proper optical theory, for in accordance with this, the image being smaller they should separate. This they do, but by the new

[2]For calling my attention to it I have to thank one of my former pupils, Prof. D. K. Shute, M.D., of this city.

arrangement in the later instruments as the image grows smaller the adjacent bands encroach one upon the other as is readily understood from the accompanying figure.

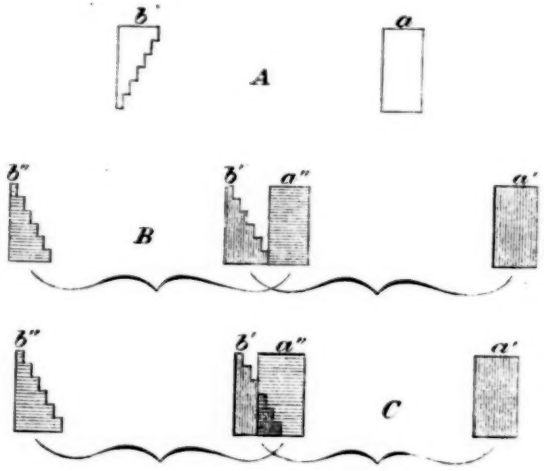

FIG. 1.

In A we have the object *a b*, the band *a*, which represents one end being rectangular, the other end *b* having gradations in steps. This is as it appears, one the arc of the ophthalmometer as seen by the examiné. When the cornea is viewed by the observer through the telescope and prism,—the arc being in a horizontal position — the two images of the corneal reflection are seen inverted in the relative positions as shown in B, where *a' b'* is one image and *a''*, *b''* is the other. The inner side of *a''* is in contact with the inner side of *b'*. The distance between the inner sides of each of these images is 3 mm. when the instrument is properly adjusted. Now let the arc be turned to the vertical meridian which is the more

strongly curved and more highly refracting. According to the optical law, the images *a'* *b'* and *a''* *b''* should be smaller, and *a'* should approach *b'* and *a''* should approach *b''*, and in doing this of course *a''* and *b'* will overlap as shown in C. And this is how it comes about that we have a crossing of the band in the meridian of shortest radius, greatest curve and highest refraction.

My mistake undoubtedly arose from the fact that I wrote the description after studying Javal's first papers, and did not notice, in reading his subsequent articles, the change he had made in the disposition of the bands. Originally he had the outer side of *b''* to be applied to the outer side of *a'* in the horizontal position which would of course be attended with a separation of *a'* and *b''* when the arc was turned to the vertical position.

The same mistake into which I have fallen has been committed in several treatises and articles written on the instrument that have appeared since the publication of my treatise.

The recently issued work of De Schweinitz has this statement on page 128: "If the image overlaps in the vertical meridian the radius of course is longer in this meridian and there is astigmatism. If the image separates with the bar vertical, this meridian has a shorter radius than the horizontal." This is, of course, exactly the opposite of the true condition of things. In his text book, Noyes, while giving the correct method of reading, offers no explanation of the principles involved and, besides, is somewhat confusing as to the exact position of the arc, since in one place he speaks of the index of the instrument indicating the position of the arc (the index being at right angles to the arc) and in another of the direction of the arc itself as if they were identical. Würdemann, in a paper published in the *Journal of the American Medical Association*, for August 27, 1892, copies my erroneous statements entire. Koller, in a paper published in the same journal for September 13, 1890, and which purports to give a full and complete description of the instrument, while stating the optical principles correctly, not only makes the same blunder

as to the practical application, but illustrates it with a drawing which is exactly the reverse of what it should be for the instruments now in use and which he figures.

It should be remembered that the only thing the ophthalmometer can do is to give data for calculating the radius of corneal curvature. It does not even give us the refraction of the cornea for this can be known accurately only when we have in addition the index of refraction of the corneal tissue[3] and the aqueous humor. In Javal's first paper (*Ann. d'ocul.*, Juil., Aout 1881), he assumed this to be 1.35 and his values for the corneal refraction there given are based on this index. He afterward took what is now accepted, I believe, as the true index, 1.337, but has not, I think, published a revised table. I have myself constructed a table of reciprocal values in refraction on these data[4] which makes them correspond to the reading on the arc of the ophthalmometers now in use.

It would seem from some recently published papers that this really valuable and necessary instrument is in danger of suffering at the hands of a few of its enthusiastic champions, who are claiming reliance upon it to an extent which certainly my experience will not justify. It will not allow us to dispense with the use of a mydriatic in all cases, nor is any one warranted in ordering cylinders in accordance with the reading of the instrument without having verified these readings by other methods. The final resort, as I have always maintained, must be lenses and test-types.

In the majority of cases the corneal astigmatism as shown by the ophthalmometer is the total astigmatism of the eye making allowance for the 0.5 D. astigmatism of the lens, con-

[3]Some one has written of the posterior surface of the cornea as a possible factor, but it should be remembered that the corneal tissue and the aqueous humor are of nearly the same index, and that irregularities of the posterior surface of the cornea could play no important part in changing the refraction.

[4]An analysis of the refraction of 576 healthy human corneæ examined with the ophthalmometer by Javal-Schiötz, "Transactions of the American Ophthalmological Society," 1888.

trary to the rule, but in an important minority it is not; and the longer I work with the instrument the more important a factor I find lenticular astigmatism to be.

The ophthalmometer is a great instrument, but like all other great things, it has its limitations, and we should not, in our admiration, allow these to be lost sight of.

SELECTIONS.

THE PRIME ETIOLOGICAL FACTOR OF GLAUCOMA IS CONSTITUTIONAL.[1]

BY S. O. RICHEY, M. D., WASHINGTON, D. C.

This paper will be occupied with the presentation of one idea, for a *resume* of the literature of glaucoma would unprofitably occupy much space, as so much has been written offering diverse views of its different features, each with a show of reason.

Mr. Jonathan Hutchison, in the Bowman Lecture, 1884, discusses the relation between certain diseases of the eye and gout. The tissues of an individual long subject to the causes of gout may become modified in such a way that they are liable to suffer in a peculiar manner when exposed to the ordinary causes of disease; the *nervous* and *vascular* systems are specially so disposed. Rheumatic gout may have such a relation to true gout, and he names "hot eyes," calcareous bands of the cornea, arthritic iritis, relapsing cyclitis, *glaucoma*, and retinitis hæmorrhagica as having such connection; and asks if it can attack any of the *structures of which the nervous system is composed.*

In the London *Lancet*, January 1873, he describes an iritis occurring at an early age, differing from other forms of arthritic iritis, in being persistent and insidious, rather than paroxysmal. Without any attack of acute inflammation, adhesions quietly form between the iris and the capsule of the lens.

[1]Read at the meeting of the American Ophthalmological Society, July 20, 1892.

* * * This affection usually begins in but one eye, and advances to almost entire loss of vision in it, before attacking the other. It is insidious, and for the most part painless, but is liable to exacerbations and periods of improvement. It is remarkably intractable, prone to attack both eyes, and to end in blindness.

Such is the position of an acute observer as to the influence of gout upon the eye.

The question as to the *cause* of increased tension is still open, whether due to the *too rapid infiltration*, or to *impeded excretion*, with a leaning to the latter.

Opposed to Mr. Priestley Smith's theory, that "glaucoma of every form is essentially a disease of retarded excretion,"[2] are the conclusions of Schnabel,[3] supported by clinical and pathological studies, that "glaucoma may be present without obliteration of the sinus of the anterior chamber; that the latter can exist without glaucoma; that glaucoma can be cured without obliteration of the sinus of the chamber being removed."

"It has been proved by Mr. Windsor,[4] of Manchester, that acute glaucoma may occur where there is congenital absence of the iris."

A doubt, which reaches almost a denial, is general as to whether excavation of the disc is due to pressure, or not. In Rydell's case, blind from acute glaucoma of three weeks' standing, *without excavation*, pain was relieved and tension reduced, but vision was not improved. Mauthner[5] claims that: "We find in the beginning of an excavation that pressure frequently is not increased. I have recently examined the left eye of a patient, in which there is the beginning of a pressure excavation, of which there was not the slightest sign a year ago, when I saw him for paresis of one of the muscles. The functional disturbance is extraordinary, and shows itself in transitory obscurations; central S. is less than in R. E. which has S=$^{6}/_{VI}$, while with L. E. a few letters of 6 are not seen at 6 m. distance. Without glasses the patient, who is forty-five years old, reads with R E., J. 2, with the left eye J.

3. F. undisturbed. The well known appearance of the vessels is very marked at the upper lateral edges of the papilla. T. is precisely the same in both eyes, and falls even below the physiological maximum. Would such a pressure produce such a picture?"

"Some morbid process has attacked the intra-ocular end of the optic nerve, causing a diminished resistance (softening) of the lamina cribrosa, so that it yields to even normal pressure in the eye, but at the same time there is going on in the optic nerve and alteration, which has the greatest resemblance to that in the lamina cribrosa, and leads to a softening, to a giving way of the supporting connective tissue."[6]

Reading these comments on glaucoma with a free mind, our previons ideas are subverted, because we must conclude that increased tension is not necessary to excavation; that excavation is not always present, even when increased tension has existed sufficiently long to produce it; that excavation may result from[7] "some morbid process" in the nerve, lessening its resistance; that increased tension is not dependent upon obstruction of the channels of excretion.

If the last proposition be true, that increased tension is *not* dependent upon obstruction of the channels of excretion—and Schnabal supports his conclusion by dissections of the organ which he had observed while affected with the malady—then increased tension *must* be caused by too rapid inflitration, or secretion. Schnabel argues further,[8] that glaucoma is a disease of the bloodvessels of the eye, which develops either gradually, or at once, in the region supplied by the long anterior and posterior ciliary arteries, the central bloodvessls, and those of the sclerotic circle; that the disturbances of nutrition and function are the direct result of these disturbances of circulation, etc.

Mr. Priestley Smith's theory was obviously derived from the study of glaucoma of local origin; and yet, as Mr. Brailey, of London,[9] says, "it fails to account for temporary glaucoma, for glaucoma without the characteristic application of the iris, for glaucoma in young persons, for one-sided glaucoma, glauco-

ma in aphakic eyes, and especially for cases where a traumatic dislocation of the lens backward has been quickly followed by increased tension. It does not, also, explain the *invariable inflammation* and *atrophy* of the ciliary body and optic nerve."

To the theory of increased secretion, or more properly too rapid infiltration, a *vis a tergo*, some derangement of the general system, is a *sine qua non*. The uric acid diathesis, of which gout is a characteristic feature in many instances, offers the most satisfactory explanation; true gout, of acute inflammatory glaucoma; rheumatic gout, of chronic simple glaucoma.

In nearly all particulars acute gout of the toe and acute inflammatory glaucoma are alike. Observe the points of resemblance. (See Table, next page).

Thus, each may be inherited and have the premonitory symptoms; the attack is sudden and at night; in each it is characterized by great pain, engorgement, and tension, followed œdema and exfoliation; duration, from a few days to a few weeks; recurrence of the affection, possibly to attack the other side, or to become chronic. No pus.

Such is the clinical picture.

That acute inflammatory glaucoma is more frequent in females, and gout of the great toe more frequent in males, may be due to the greater emotional tendencies of women; for, according to Schweigger "mental emotion and loss of sleep favor acute glaucoma."

While women derive a certain immunity from podagra by reason of menstruation (Hippocrates), yet at the approach of the climacteric, a period of greater or less tendency to vascular cerebral disturbance, arising from the intermittence of the derivative action of this function, acute inflammatory glaucoma is most frequent, and chronic simple glaucoma develops.

"The great toe[10] contains a considerable amount of tissues peculiarly liable to become the seat of the deposition of urate of soda; as, for example, the cartilages and ligaments, tissues having either little vascularity, or nourished independently of bloodvessels; the great toe being very remote from the heart,

TABLE.

ACUTE INFLAMMATORY GLAUCOMA.	ACUTE GOUT OF THE GREAT TOE.
1. An inherited tendency.	1. An inherited tendency.
2. Most frequent after the period of presbyopia.	2. Most frequent after the beginning of senile changes.
3. First attack is usually in cold weather.	3. First attack, usually in winter, or spring.
4. Premonitory symptoms: Impaired A.; premature presbyopia, increased H.; halo, rising clouds or smoke, heaviness of brow, shooting pains in the eye, increased tension. These may be so slight as to cause no anxiety.	4. Premonitory symptoms may be so slight as to pass unnoticed, or may be very distressing.
5. Sudden seizure, usually at night.	5. Attack is sudden, usually between two and five o'clock in the morning. (Garrod).
6. Constitutional disturbances; febrile excitement, with some nausea and vomiting.	6. Chilliness, heat of skin and perspiration, thirst, loss of appetite, a white tongue, constipation, and restlessness.
7. Circumorbital pain, peri-corneal and sub-conjunctival injection, slight protusion of globe, sluggish, dilated iris; cornea dull and anæsthetic, humors greenish, ischæmia.	7. Toe is swollen, red, hot, and exquisitely tender. Veins proceeding from the toe are turgid with blood, and the joint is stiff. Great tension of the skin.
8. As the attack passes off there is great chemosis, lachrymation, and photophobia. The cornea becomes roughened.	8. As the attack passes off there is pitting of the skin (œdema), then desquamation.
9. The inflammatory attack passes off in a few days or weeks.	9. Duration, from four days to three weeks.
10. The disease is not arrested: there may be a recurrence of acute inflammatory attacks, chronic inflammatory exacerbations, or the disease may progress insidiously.	10. Gout recurs, and the frequency of the paroxysms increases.
11. No pus.	11. No pus.
12. Urine. ?	12. Urine scanty, high-colored, and deposits a colored sediment on cooling.
13. No analysis of aqueous humor, so far as I know.	13. Synovia contains urate of soda.
14. The disease may attack first one eye and then the other.	14. Gout not uncommonly seizes first one great toe, then the other.
15. Occurs most frequently in women.	15. Is rare in women.

the circulation is weaker there. * * * The reasons for the great toe on one side of the body being affected apply equally to the other; and hence, the disease not uncommonly attacks first one toe and then the other, within the short space of a few hours or days."

Anatomically, the eye is an extremity of the body, not quite so far from the heart as the toe, and is exposed to variation of temperature and to injury; the sclerotic, the cornea, and the tendons of the extrinsic muscles are of dense fibrous tissue, with little vascularity; the stroma of the choroid and iris is of reticular connective tissue, supporting pigment cells, blood-vessels, etc., the zonule of Zinn is a *fibrous* perforated membrane, the lense capsule is a structureless membrane, the corpus vitreum depends upon bloodvessels not its own for nutrition, and contains mucin, and (Picard) 0.55 per cent. of urea, and about 0.75 per cent. of sodic chloride. The posterior surface of the iris and ciliary body secrete the aqueous humor (synovia?) which contains a small amount of albumin, sugar, and sodic chloride, equal to $^1/_{50}$ of its volume.

With increase of blood-pressure and intra-ocular pressure, there is increase of albumin and the production of fibrin in the anterior chamber. (Jessner and Grünhagen.)

Taken with the fact that a local derangement, as a dislocated lens, does not seem sufficient to cause the *whole* train of symptoms, general as well as local, called glaucoma (though it may precipitate an attack which would probably have taken place at the latter date), the clinical history of a seizure and the anatomical peculiarities of the regions under consideration present a picture of such mimicry as we find nowhere else repeated. The crucial test, the presence of urate of soda, I have had no opportunity to apply since recognizing the resemblance.

To again read Garrod,[11] "The impure state of the blood, due the presence of urate of soda, is probably the cause of the disturbance which often precedes the gouty paroxysm; that is; of the so-called premonitory symptoms. Urate of soda in abnormal quantity in the blood is essential to an at-

tack of gout, * * * but does not constitute gout; * * * that the amount of deposited urate of soda is not in porportion to the intensity of the inflammation, and that in some the infiltration may ensue and give rise to scarcely any inflammatory action. * * * The inflammation of the gouty paroxysm tends to the destruction of the urate of the soda in the blood of the inflamed part, and probably of the salt also which is thrown out." Soelberg Wells[12] observes that "males who are attacked by glaucoma frequently suffer from gout, or disorders of the digestive organs;" of primary glaucoma, "when once the one eye has become affected with glaucoma there is great tendency in the disease to invade the other also."

Mr. Hutchinson[13] asserts that "all forms of rheumatism, and all forms of gout, are included in the common term, arthritic. But we cannot limit the term to the joints, as its etymology might seem to require, but must allow it also to apply to certain affections of the muscles, fasciæ, tendons and other fibrous structures which have been proved to be dependent upon the same peculiar state of health. * * * Under the term rheumatism we include all arthritic maladies which are not proved to be gouty. * * * I must protest, at once, against any attempt to limit the term gout to cases in which attacks of acute inflammation of the great toe occur. * * * Rheumatism differs from gout in being of *nerve* origin, and due to reflex disturbance of nutrition; * * * it is, according to my hypothesis, the basic diathesis to which a small minority of cases of gout is superadded."

The younger Garrod says that rheumatic gout lacks the distinguishing feature of gout, urate of soda.

Many of the manifestations of rheumatic gout are associated with chronic glaucoma. viz.: enlarged or distorted joints, a peculiar senile pallor, or muddiness of the skin; periods of mental depression, and other symptoms, attributable only to changes in the nervous system. I have found nowhere any reference to pathological alteration of nerve tissue in gout although the existence, character, and specific cause of such, changes, which are *presumed* to exist because of the nervous

symptoms present in lithiasis, would have an important bearing upon the subject in hand, as explaining the structural changes in the lamina cribrosa and the intra-ocular end of the optic nerve, the condition of diminished resistance associated with excavation without increase of tension, in cases of chronic glaucoma.

Dr. W. W. Johnston,[14] Washington, D. C.. published some thoughts "On the Nature and Treatment of Forms of Disease characterized by Indigestion, the Presence of Bile, Urates, and Uric Acid in the Urine, and by Nervous Symptoms," which suggest a possible cause and explanation of the nerve changes in chronic glaucoma. In his own words, "The question of the continuous production of toxic substances in the intestinal canal in health, and the protection of the organism by the absorption of poisons in alterations of the gastro-intestinal tract, was developed in detail by Professors Albertoni and Silvia at the meeting of the Fourth Italian Congress of Internal Medicine, held in Rome. Professor Silvia enumerates the following substances as probable poisons; peptoxine bases (ptomaines and leucomaines), idol, phenol, lactic acid, ammonia, sulphuretted hydrogen, acetone, etc. The direct proof of the fact that the nervous phenomena in such cases are due to the absorption of toxic matters from the intestines is not yet found, but the argument is a focible one. The existence of indigestion is known by the symptoms; the presence of toxic matters in the intestine in health is proved. * * * The relationship of acute indigestion and nervous disturbances, and the association of fermentative dyspepsia with nervous symptoms, and an excess of these products in the urine and faeces, give sufficient grounds for adopting this theory as reasonable."

Dr. Johnston has give much attention to the subject of digestion, and if a reference to his able paper will induce those who have the care of cases of chronic glaucoma to read it, it will probably divert attention from glaucoma, except as a local manifestation of a general malady (although he does not refer to glaucoma), broaden the view of the subject, and en-

able us to comprehend the changes in nerve tissue going on elsewhere in the system in rheumatic gout, by that which takes place in the intra-ocular end of the optic nerve, exposed to observation, in chronic glaucoma.

Returning to the subject of intra-ocular tension, Mr. Priestley Smith[15] claims that "high tension depends more upon an excess of blood in the eye than upon the excess of intra-ocular fluid," while Dr. Spender[16] has observed, as early symptoms of arthritis, increase of pulse rate with high arterial tension.

Mr. Hutchinson[17] concludes that "it is probable that there are many different forms of inflammation of the eye, or of parts of it, which are in connection with gout. They may be divided into groups: *a*, those which go with acquired, humoral, or renal gout; *b*, those which depend upon inheritance of structures damaged, or, at any rate specialized, by gout in predecessors. The difference between the two classes of affections is very marked. In the one, attacks of a transitory nature are the rule, and the attacks are often acute and attended by much pain. In the second group, although a tendency to temporary recovery and recurrence is often observed, yet, there is a great proneness to chronicity, and persistence. The invasion is often insidious, but the disease is usually in the end destructive."

If the difference between the forms of acute inflammatory and chronic simple glaucoma had been in the mind of Mr. Hutchinson the description could not have been more effective than in the specification of the two groups named above.

His address will bear reading with this thought.

Ordinarily, when both eyes are attacked by the same disease process, we rationally conclude that the cause is constitutional, and do not treat an expression of the dyscrasia, but rather its cause.

In chronic glaucoma, a local manifestation is treated (for, sooner or later, both eyes are attacked), and then we wait to see what "turns up," with about the results presented by Dr. Bull,[18] of New York, to the American Ophthalmological Society, in 1889; in the detailed history of ninety cases of chronic

simple glaucoma, subjected to the operation of iridectomy, during a period of seventeen years. The paper is most interesting and instructive, especially the summing up: "One hundred and fifty-four operations were done on the one hundred and eighty eyes under consideration. Vision was temporarily improved by iridectomy in both eyes in two cases, and one eye in six cases; but in all eight cases, after a few months, a steady loss of vision and narrowing of the field set in, and continued progressively as long as the patients were under observation.

"Vision remained unchanged, neither better nor worse, after the operation, for a period of one year or longer, in both eyes in eight cases, and in one eye in twenty cases.

"Vision grew slowly and steadily worse after the operation, in both eyes in forty cases, and in one eye in twenty-nine cases.

"Vision grew rapidly worse after the operation, in both eyes in two cases, and in one eye in eight cases."

He concludes that "the health and age of the patient exert a decided influence upon the operation, and any marked evidence of senility ie distinctly unfavorable to the operation."

Dr. Gruening,[19] of New York: "In cases of chronic glaucoma with degenerative changes, neither iridectomy nor anterior sclerotomy will give the patient the desired relief; posterior sclerotomy *may do it at times.*"

Mr. Power,[20] of London: "In cases of chronic glaucoma no operation is of much service." This terse statement, it seems to me, covers the whole ground.

The good results of operation in chronic glaucoma are in comparatively small ratio, and are therefore accidental, and not scientific; for it often precipitates disaster by additional irritation. So long as the two chief clinical characteristics of glaucoma, increase of tension and excavation or the disc, are not satisfactorily explained, the management of such cases must be empirical. The author of iridectomy for glaucoma acknowledged it to be empirical, and only experience has taught us in what cases it is of most value, those of acute in-

flammatory glaucoma; for here it saves the eye until another time; it does not cure the disease. Dr. Bull's statistics do not teach us to do iridectomy in chronic glaucoma, cases of which form of the disease are in excess of any other, unless upon the plea of *dernier ressort*—because we know of nothing better. They indicate that the majority of eyes are worse after an operation; in a few the *status quo ante* is maintained; in a still smaller percentage there is some improvement. With this diversity of result, who, save in the occasional case of *immediate* gain, or loss, to the eye, can say what influence is attributable to operating? Might the case not have done just as well without interference? Is the surgeon justified in a feeling of certainty that he he has done a service? If all such cases followed *approximately* a given course he would have a guide; but they vary so much. If it progresses slowly after an operation, it might have done so without it. If it remains stationary for a time can that be attributed to operation? If the patient goes rapidly blind, has he a right to reproach the surgeon? In operation is *possibility*, not *probability*. In simple glaucoma it has a questionable rationale, and experience teaches that, if done at all it must be done with cautio . It is double-edged, and may cut either way.

It is a prime necessity that a quiet, healthy, out-door life should be led, apart from occupations of much nervous excitement, causing loss of reserve force; that a condition of self-possession should be maintained; that the dietary should be regulated as to time, quantity, and quality; for over-feeding and bad feeding is a conspicuous vice of the age. In adult life the effort should be to preserve the balance between waste and repair, and to see that both processes are normal. This is a duty which the family physician may share.

As such cases pursue so chronic a course, it would seem wise to discover the constitutional cause, and to begin with that, instead of with the last expression of the disease, leaving the cause in action.

Rational management of the disease involves a study of the general condition and a correction of all the habits of the

individual. This is difficult, but our function is advisory, and each sufferer must "work out his own salvation" with our guidance.

By controlling the quantity of food productive of uric acid and by reducing the whold quantity to the possibility of easy digestion and assimilation, thus lessening the amount of toxic substances in the intestinal tract; by the regular entire excretion of what is excessive by way of the kidneys and bowels, harm in this way is obviated. Tonic aperients (not irritants), which encourage natural action of the intestines, serve a good purpose when used with judgment. Hunyadi water, taken at bedtime, lies in the track all night, does not purge, but by its solvent power prevents accretions. Nothing should be done to lessen the digestive power, and a quantity of food should be taken, small enough to *insure* its digestion and proper disposal. Anything (as coffee) which retards digestion must be rejected for obvious reasons.

Salicylate of phenol, it is claimed, has been found in the joints of gouty persons taking it; therefore its purpose is apparent.

Lithia waters secure the excretion of some uric acid; piperazine, a new synthetical compound, is recommended as having twelve times the solvent power of lithia upon uric acid. Strychnine acts by stimulating the functional activity of all the organs of the physical economy.

Galvanism, if properly and steadily used, is profitable. After ten minutes' use of two milliampères direct current to the sympathetic, in an ordinary case, ocular tension in lessened the pupils seem more active, and the patient becomes calm, often almost falls sleep. By the experiments of Onimus and Legros[21] it has been shown that if the direct current (positive pole at the nerve center) be employed, the circulation is augmented; within a few moments the arteries have increased in bulk, and the whole network of capillaries is seen in great commotion. Faradization contracted the bloodvessels, but after a time contraction ceased, and the arteries became larger than before the application. The continuous current, on the

other hand, renders circulation more active, and reestablishes it when it has been arrested. The induced current produces spasmodic contraction of the unstriped muscle, while the continuous current produces a *vermicular* contraction (Bartholow). The latter thus favors the natural movement of the vessel, and while *directly* increasing the amount of blood passing, by reaction the amount of blood in the part supplied by the vessel is reduced to the normal. The object to be gained, stimulation of the cercival ganglia, the trophic centers of the region of the trigeminus, is accomplished as well with the cathode held in the hand as in contact with the affected region; yet, when placed on the temple, or above the eye, it has some additional *mental* effect, which is not undesirable.

The writer has endeavored to cover the ground as concisely as possible: to offer the salient points of a view of the subject he has entertained for several years, especially in regard to *too much food.* He thinks that in the hypothesis discussed we find the true etiological factor of the most intractable of diseases, chronic glaucoma; that acute inflammatory glaucoma is a poroxysmal expression of the same affection; that local irritation, or trauma, excites an attack of glaucoma only in the presence of the dyscrasia; that operation saves the eye during a paroxysm; that operation serves little purpose in chronic glaucoma, even when it does not, by irritation, hasten the disease process or precipitate a paroxysm; that chronic glaucoma is a neurosis—progressive atrophy with the feature of inflammation with deficient power, varied by periods of *seeming* rest; that correcting and controlling individual habits, especially in the *amount* and character of food taken, will do more to preserve vision than operation; add that there may be a possibility of aborting chronic glaucoma, if the tendency to it be recognized at an early stage.

LITERATURE.

[2]Trans. Seventh International Medical Congress, vol. iii, p. 84.

[3]Archiv. Ophthalmol., vol. vii, p. 14.

[4]A Practical Treatise on Diseases of the Eye, by Haynes Walton, London edition, p. 1170. See Ophthalmic Review.

[5]Von Graefe's Archiv, 1872, vol. xviii, pp. 1-51.

[6]Archiv Ophthalmol., vol. viii, p. 38.

[7]Vide supra, p. 39.

[8]Garrod, on "Rheumatoid Arthritis," Reynold's System of Medicine, p. 553; "In the early stage, when swelling is prominent, a considerable increase of synovial fluid is found, and the joint exhibits the same appearance as in case of ordinary inflammation. The lining membrane is often red from over-injection of the blood-vessels. If the bone is sawn through, it is often found spongy, and contains a large amount of *oily water*, from the occurrence of a *species of fatty degeneration*. N.B. All italics are my own.

[9]Archiv f. Augenheilkunde, vol. xv, p. 311.

[10]Trans. Seventh International Congress, vol. iii.

[11]Reynolds' System of Medicine, vol. i, p. 533.

[12]A Treatise on Diseases of the Eye, 3d Amer. ed., 1889, p. 589.

[13]Trans. Seventh International Medical Congress, vol. ii, p. 92.

[14]The Medical News, March 12, 1892.

[15]Ophth. Rev. vol. vi, p. 196.

[16]Garrod: A Treatise on Rheumatism. Am. ed., 1890, p. 245.

[17]Ophth. Rev., vol. iii, p. 385.

[18]Trans. Amer. Ophth. Soc., vol. vi. part 2, pp. 246, 291.

[19]Ibid., 1889.

[20]Trans. Seventh Internat. Med. Congress, vol. iii, p. 106.

[21] raité d'Electrode Medicale, Paris, 1872.

CONTRIBUTION TO THE SUBJECT OF INTRACRANIAL LESIONS WITH DEFECTS IN THE VISUAL FIELDS.—FIVE CASES WITH AUTOPSIES.

BY CHARLES STEDMAN BULL, M. D.,

Professor of Opthalmology in the University of the City of New York.

CASE I.—PACHYMENINGITIS OF THE CONVEXITY OF THE BRAIN, WITH EXTENSIVE ENDARTERITIS AT THE BASE.—A lady, aged fifty, presented herself in May, 1886, with the following history: Four years before, she had suffered with a very severe mental shock, brought on by the sudden death by an accident of two members of her immediate family. Previous to this she had always enjoyed a fair state of health, though she had never been strong. The sudden shock caused a series of convulsions, which ended in a profound condition of neurasthenia, lasting for more than five months before she began to improve. The nervous prostration was accompanied by a profuse menstrual flow, coming on every three weeks and lasting for twelve days. Somewhat less than a year later the vision of the left eye became affected, the first symptom being night-blindness. The amblyopia of the left eye progressed very rapidly, so that in less than three months she was unable to read the largest type with any glasses. With every recurrence of the uterine hæmorrhage the vision in the left eye became suddenly much worse. About a year ago the right eye became affected the same way, but not with the same rapidity, the first symptom being again night-blindness. She then began to suffer from headaches of a peculiar type, beginning at the vertex with a feeling as if a sharp instrument had been

driven through the skull, and had then been turned round in the brain, and she would shriek with the sudden pain. When I saw her in 1885 these headaches had changed in character and were of a dull, persistent nature and located in the occipital region. At that time the motility of the eyes was unimpaired, the irides and pupils were normal, and the media were clear. An examination of the eyes gave the following results: R. E., $^{20}/_{XXX}$, unimproved with glasses. Reads Jaeger 4 with sph. +.D. 1.50. Small positive central scotoma for form and color. Color sense normal outside the limits of the scotoma. Opthalmoscopic examination negative. L. E., $^{2}/_{CC}$ eccentrically. Large irregular central scotoma. Total color-blindness. Neuroretinitis in the stage of decline, but without hæmorrhages or positive exudation and without papillitis. Optic nerve in the first stage of atrophy, with slight discoloration of the disc, and with the arteries and veins reduced in caliber. At times there is entire obscuration of the field of vision of the right eye, which always occurs slowly and as slowly disappears. The hearing was normal, the knee-jerks were normal, there was no difficulty in walking, and the dynamometer gave a fairly normal result. The urine was repeatedly examined, and showed nothing abnormal, save an excess of urates.

Under observation the vision in the left eye gradually sank to distinguishing the movements of the hand eccentrically, and the vision of the right eye diminished to $^{20}/_{LXX}$. The left optic disc became atrophied with indistinct outline. The patient was seen at intervals up to the spring of 1890, when she died. The headaches gradually returned with increasing severity and frequency, and she became at times mildly delirious. During the last year of her life she had repeated attacks of unilateral convulsions, mainly confined to the left side, but occurring occasionally on both sides, and she died in an unusually violent convulsion, which seemed to be general in character. This patient had never had syphilis or any symptom of tuberculosis, though the latter disease existed in her family.

The autopsy showed extensive pachymeningitis of the convexity of the left cerebral hemisphere, most marked over the

anterior lobe, with some patches over the anterior lobe of the right hemisphere, and one large patch, the size of a fifty-cent piece, over the parietal lobe of the right side, low down. All the arteries at the base of the brain were thickened and their lumen was narrowed, and this was particularly marked in the left anterior and middle cerebral arteries. The optic tracts and chiasm presented no microscopic changes. There was no exudation and no pachymeningitis at the base of the brain. There was no extravasation of blood anywhere within the brain.

The relation of cause and effect in this chain of symptoms seems difficult to unravel. We have to deal, first of all, with a sudden and violent mental shock, followed immediately by convulsions, and ending in profound nervous prostration. On recovering from this latter condition, menorrhagia set in, and was followed by night-blindness of one eye and gradually increasing loss of vision, with both subjective and objective central scotoma for form and color. Headaches then began, which were at first boring in character and located at the vertex, but subsequently became of a dull, persistent character, and were centered in the occipital region. Then the second eye became affected in the same way, while the first eye developed a neuro-retinitis which ended in atrophy. Then followed unilateral convulsions, transient attacks of amblyopia in the second eye, and, finally, a general convulsion, ending in death. Reasoning from the results of the autopsy, the endarteritis was probably already well developed at the time of the occurrence of the mental shock, which, in its turn, hastened the progress of the arterial degeneration and indirectly the development of the pachymeningitis. The neuro-retinitis was probably to be attributed more to the long-continued loss of blood than to the pachymeningitis of the convexity, for, though there was marked disease of the arteries at the base of the brain, there was no meningeal complication in this region. The occurrence of night-blindness as the first symptom of the loss of sight was unusual, as there was no extensive retinal lesion in the fundus, and none at all at the periphery. The

scotoma in the field of the right eye gradually increased in diameter until it reached 45° on the nasal side, 60° on the temporal side, 35° upward and 40° downward. The convulsions were probably the result of the vascular degeneration and the meningeal inflammation.

CASE II.—SARCOMA OF THE OPTIC CHIASM AND NERVES.—A young man, aged twenty-four, called on me in February, 1888, and gave the following history: For more than a year he had suffered from headaches, which were at first confined to the frontal region and were slight and transient. They, however, increased somewhat rapidly in severity and intensity, and involved the whole head; so that at times he felt as if his skull would burst. After about six months had passed there appeared muscular twitchings in the upper extremities and face, and occasional attacks of vertigo, but with no loss of consciousness. About the same time the vision of both eyes also became affected, as if a slight haze covered everything, and this gradually grew worse. Three weeks before I saw him, while he was in the third story of an unfurnished building superintending some work, a very violent headache came on accompanied by vertigo, and this was succeeded by a general convulsion, in which he fell to the ground, a distance of thirty feet, striking on his back and side. He was unconscious for a few minutes and then came to his senses, and after a while stood up and walked home, a distance of nearly half a mile, without assistance. This was his first convulsion and there has been none since. The headaches became constant and were at times very severe. An examination revealed nothing abnormal in the appearance or motility of the eyes. The irides and pupils were normal and the media clear. The optic nerves were very hyperæmic and the veins pulsated, but the outlines of the papillæ were clearly defined and the retinæ were intact. There was no diplopia. The field of vision showed a slight concentric narrowing in each eye. Vision was $^{20}/_{c}$ in both eyes. The hearing was normal, and there was no tinnitus. The patient had never had syphilis, and appeared to be

in a good state of health. The urine was carefully and repeatedly examined, but nothing abnormal was found except a high specific gravity. It seemed impossible to make a satisfactory diagnosis, though, from the persistence and severity of the headaches, the muscular twitchings, and the convulsion, the presence of a tumor was suspected. Before I saw the patient he had had four partial tenotomies done for the relief of his headaches—three on his right eye and one on the left—but with no result. His refraction was hypermetropic, D. 0.75 in each eye under atropine. He had no astigmatism. Potassium Iodide was administered, and he was requested to report at the office once a week, which he did faithfully as long as he was physically able to do so. The vision slowly grew worse, and the concentric limitation of the fields gradually increased. The headaches continued in spite of the large doses of potassium iodide (sixty grains three times a day), and after two months it was discontinued, as he began to show signs of iodism. About three months after I first saw him the optic discs lost their hyperæmic condition and began to grow pale. Strychnine was then administered, but he was obliged to discontinue it, as it made his headache worse. Nearly five months to a day after I first saw him he had a violent convulsion, lasting nearly six minutes, and this was followed by a second some hours later, after which he never had another. The optic disc rapidly assumed an atrophic condition, the field grew very narrow in both eyes, and vision sank to $^{2}/_{cc}$. He became very irritable, and this condition was followed very soon by a stupid, somnolent state, which gradually deepened into profound coma, in which he lay for nearly ten days before death came—about eight months after his first visit. The autopsy revealed nothing abnormal on the convexity of the brain, but the skull in the vicinity of the left fronto-parietal suture was very much thickened, and the dura mater was very firmly adherent to it. On attempting to remove the brain from the skull, a growth was apparently discovered at the base in the vicinity of the sella turcica. After much careful dissecting in the vicinity with the handle of the scalpel and the

finger, and division of the spinal cord as low down as it could be reached, the brain was removed, and it then became possible to study the location of the tumor. It was as large as a Brazil-nut, and involved very closely the optic chiasm, both optic nerves near the chiasm, and the hypophysis. It was moderately hard, with a smoth surface, and, on being divided, was seen to be of a grayish hue and of the same consistence throughout. The optic nerves just beyond the chiasm were flattened by the pressure of the tumor. The growth did not seem to extend backward into the optic tracts, nor upward into the hemisphere, but it had made a distinct depression in the under surface of both hemispheres. It was somewhat firmly adherent to the dura mater at the base. Macroscopically the tumor had apparently originated in the optic chiasm or in the connective tissue surrounding it. There were no other lesions discoverable anywhere in the brain after a most minute examination had been made. The tumor was carefully hardened and then examined microscopically. It proved to be a small cell sarcoma, tolerably vascular in character, with relatively great development of the connective-tissue framework. It could not be accurately determined whether it had originated in the hypophysis or in the connective tissue of the chiasm. The nerve-fibers were in many places entirely atrophied, and this was particularly noticeable in the origins of the optic nerves. There were no signs of meningitis or of neuritis, and the case seemed to be one of simple atrophy from compression. Papillitis or choked disc was conspicuous by its absence, which was an interesting point in the case. Another interesting fact was the very small number of convulsions which occurred in the course of the development of the tumor, and the long period—nearly six months—which elapsed between the first and second convulsions. The autopsy showed that no injury to the skull had been caused by the severe fall of thirty feet, and this corroborated the statement made by witnesses of the accident that he had struck on his back and side, and not on his head. No satisfactory attempt was made to locate the tumor before death, and not the slightest suspi-

cion was entertained that the growth involved the optic chiasm.

CASE III.—SARCOMA OF THE LEFT OCCIPITAL LOBE OF THE BRAIN; BILATERAL RIGHT HEMIANOPSIA.—In December, 1888, a gentleman, aged thirty-seven years, presented himself at my office with the following history: For the past seven months he had noticed a loss of vision in the right half of each field. For about two months previous to the appearance of the hemianopsia there had been a constant severe headache in the left parietal and occipital regions, but this pain gradually grew less and finally subsided. Six weeks ago he suddenly lost completely the sense of smell. For the last three weeks he had noticed a failure of vision in the remaining portion of the field of the left eye. Examination showed nothing abnormal in the external appearance of either eye, and the motility of both eyes was unimpaired. R. E. $=^{20}/_{C}$, with sph.—D. 1 ⌒ cyl.—D. 1.50 axis 180° $=^{20}/_{L}$. L. E. $-^{20}/_{LXX}$, with cyl.—D. 1.50 axis 180° $=^{20}/_{XL}$.

The media were clear. Both optic discs were pale, the discoloration being most marked on the temporal side and in the left eye. There was a slight reduction in the caliber of the retinal arteries. The perimeter showed a bilateral right hemianopsia, with some concentric limitation in the remaining half of the field in each eye. At the time I first saw the patient there were no other symptoms than those already mentioned. He had contracted syphilis twelve years before, the chancre being followed by secondary symptoms, but he had been entirely free from constitutional symptoms for more than six years until his headaches appeared, and these were confined to the left side. No explanation could be offered for the recent sudden onset of the anosmia, which lasted till the death of the patient. There was no interference with the sense of hearing, and nothing abnormal in the appearance of the drum-heads. A careful rhinoscopic examination showed nothing but a mild form of chronic naso-pharangeal catarrh, with some hypertrophy of the adenoid tissue in the naso-pharynx. In spite of

the absence of other symptoms. the hemianopsia and the beginning atrophy of the optic discs pointed to the existence of a lesion in the brain on the left side, and probably somewhere in the vicinity of the cuneus. With the patient's syphilitic history, it was supposable that the intracranial lesion was a gumma, and on that supposition potassium iodide was given for a period of nearly three months, the dose being increased until he took six drachms daily, which he bore very well. But, instead of there being any inprovement, there was a progressive loss of vision and a steady increase in the concentric limitation of the field of both eyes. About two months after he came under my observation the headaches returned in the occipital region, and finally became continuous, although they were never very severe. The potassium iodide was then stopped as useless, and the only treatment consisted in relieving the patient's symptoms as they arose. There was never any hemiplegia or hemianæsthesia and no symptoms of motor disturbance till two weeks before his death, when he began to have muscular twitchings of the face and hands, which never amounted to an actual convulsion. He gradually became stupid, lost his memory, sank into coma, from which he at first could be aroused but which soon became profound, and in this condition he died, not quite fourteen months after the first occurrence of his headaches.

The autopsy proved the diagnosis of an intracranial tumor and its location to have been correct, but the microscopic examination showed that it was not a gumma. The tumor, the size of a large walnut, or rather olive, was found in the cuneus on the left side. It lay close to the median line and near the base of the left occipital lobe, its long diameter pointing to the left side. It was of firm consistence, perfectly smooth, apparently inclosed in a capsule, and the surrounding brain tissue seemed to the naked eye normal. There was no other lesion found in the brain. Careful examination was made of the vicinity of the olfactory nerves, but there was no sign of any inflammatory or softening process. The olfactory lobes were atrophied and the olfactory nerves reduced to mere threads.

Not a trace of meningeal or periosteal inflammation could be found anywhere within the skull. An examination of the tumor showed it to be a typical example of the small-cell sarcoma, with marked development of connective-tissue trabeculæ. In this patient the pulse, respiration and temperature were closely watched, but there was nothing abnormal observed till toward the end, when there was a slight evening rise in the temperature.

CASE IV.—THROMBOSIS IN THE MIDDLE CEREBRAL ARTERY; RECENT CLOT IN THE MIDDLE CEREBRAL LOBE; SARCOMA OF THE RIGHT OPTIC TRACT; BILATERAL LEFT HEMIANOPSIA.—Early in October, 1889, an old lady, aged seventy-two years, was brought to me by her son, who gave the following history: About six months before, having gone to bed one night as well as usual, she was awakened early next morning by a severe pain in her head, a sense of confusion, and apparently total blindness in the left eye. She had previously been in fair health, but close questioning brought out the fact that she had suffered from headaches at intervals for more than a year, which she had attributed to some abnormal condition of her stomach. She was confined to her bed several weeks, complaining all the time of the blindness, confusion of ideas, and a numbness of her right arm and leg. She slowly recovered from most of these symptoms, but ever since she has been blind on the left side in both eyes.

An examination showed the following condition: Slight ptosis of both upper lids, but no diplopia. Speech still thick and slow, as if she were searching for a word. The tongue pointed to the left side. Partial right facial paralysis. R. E. = $^{20}/_{XXX}$. L. E. = $^{20}/_{XL}$, unimproved. Irides and pupils normal. Slight peripheral and nuclear opacities in both lenses. Some small floating opacities in the vitreous of both eyes. Ophthalmoscopic examination negative. The perimeter showed typical bilateral left hemianopsia, and in addition concentric limitation of the halves of the visual fields still remaining. There was organic valvular disease of the heart,

with aortic obstructive murmur and hypertrophy of the organ. Several exhaustive analyses of the urine gave negative results.

In going carefully over the history of the case, I thought the patient had probably had a thrombosis and subsequent rupture of the middle cerebral artery on the left side; and, from the condition of the heart and blood-vessels, I gave an unfavorable prognosis and an opinion that she would probably die in another similar attack at no distant day. She lived, however, for nearly twenty months, during which period the fields remained practically the same. The vision, however, slowly failed, which may have been partially due to the growth of the cataracts. The bilateral left hemianopsia,however, together with the headaches existing for nearly a year before the sudden attack of thrombosis, aroused a suspicion that there might be an intracranial tumor, and she was carefully watched until the end came, without discovering any additional symptom. There had never been any loss of motion in the extremities, and the ill-defined right hemi-anæsthesia entirely disappeared. She was found one morning unconscious and breathing stertorously, and remained comatose until the end, three days later, April 29, 1891.

The autopsy revealed a very interesting condition of things in the brain. In the left anterior lobe of the cerebrum was a patch of softening as large os a horse-chestnut, and in a branch of the middle cerebral artery running through it there was an old plug which obliterated its lumen entirely. There was a recent rupture of a large branch of the middle cerebral artery on the left side, and a large clot of blood in the middle lobe of the brain, close to the fissure of Sylvius. All the arteries of the brain were diseased, and some of them extensively so. At the base of the brain on the right side, overlying the right optic tract and pressing upon it, was a small tumor about the size of a hazel-nut, situated just in front of the corpus geniculatum laterale but not pressing upon it. The tumor seemed to be developed in the right optic tract, which it partially surrounded and compressed. It was of firm consistence and

smooth surface, and proved on examination to be a small-cell sarcoma, originating in the optic tract itself. Here was the probable cause of the hemianopsia and of the headaches which preceded the attack of cerebral thrombosis by nearly a year. It seems strange that there was no opthalmoscopic sign of cerebral disease such as neuro-retinitis or papillitis when I first examined the patient, but these may have appeared later, when the advancing opacity of the lenses prevented further ophthalmoscopic investigations.

CASE V. GLIO-SARCOMA OF THE CEREBELLUM.—A gentleman, aged thirty-five, called on me in February, 1890, and gave the following history: He had been perfectly well up to about three years before, when he began to suffer from frontal headaches. He had been an overworked man for many years, confined for long hours to office work, and devoting the evenings and late into the night to professional studies. His general health had always been exceptionally good, but the years of overwork and great strain had told upon his strength, and the headaches gradually increased in frequency and severity. At first confined to the frontal region. they subsequently extended all over the head. They were in the beginning intermittent, and were occasionally accompanied by nausea and vertigo. For the past three months they had been constant and at times very severe, but the nausea had disappeared. Four years ago he had received a severe blow on the left side of the head from a falling wooden shutter, which knocked him down but did not cause unconsciousness. There was bleeding from the left ear at the time, which, however, soon stopped, and was not followed by any purulent discharge. Tinnitus began in this ear at once, and the hearing was impaired and steadily grew worse. About three months before I saw him tinnitus began in the right ear and has continued ever since, but the hearing of this ear is not impaired. His condition did not vary much, with the exception that the headaches increased in frequency and severity, until about nine months before I saw him, when he suddenly began to see double. This diplo-

pia was at first accompanied by transient attacks of blurred vision, which later became permanent and progressive. By the advice of friends he consulted an oculist, who told him he was astigmatic and had extreme hyperphoria and esophoria, and that he must have the muscles or his eyes divided and wear glasses. He became a victim of the partial-tenotomy craze to the exient of five operations, without receiving any benefit. The diplopia increased his vertigo and general unsteadiness of gait, so that he was afraid to go alone in the street, especially at night. About six weeks before I saw him he first noticed a loss of sensation on the left side of his mouth, pharynx, tongue and lips, and this still remained. When he presented himself to me he had an anxious, worried expression, and a visible convergence of both eyes, from paresis of both external recti muscles. The paralysis was not complete, as both eyes could be moved outward toward the external canthi. R. E. $^{20}/_{C}$. L. E. $^{20}/_{L}$, unimproved. Irides normal in reaction and pupils of natural size. Media clear. The fundus of each eye showed marked papillitis, with numerous hæmorrhages, all the symptoms being more marked in the right eye. Homonymous diplopia for all distances. The perimeter showed and irregular central scotoma for color, but not for form. The patelar tendon reflex was normal, and the dynamometer showed no difference between the two sides of the body, and no apparent loss of power. The patient had never had syphilis. Repeated examinations of the urine showed albumen, but no casts. When he walked his vertigo was at times so marked that he staggered and apparently rotated toward the right side, and this was not materially lessened by closing on eye. At other times he apparently had no vertigo, and he walked perfectly straight when one eye was excluded.

A diagnosis was made of intracranial tumor, probably located in the occipital lobe or in the cerebellum. The patient was under observation at brief intervals from February, 1890, till his death in April, 1891. His vision slowly grew worse, until he could only recognize the movements of the hand.

There was no very marked change in the fundus, the papillitis remaining at about the same stage throughout, with the occurrence of fresh hæmorrhages at intervals. The headaches became frightful in their intensity, and could only be controlled by large doses of morphine. Toward the end he was at times wildly delirious, and about a week before his death he sank into a stupor which rapidly deepened into profound coma from which he never rallied.

The autopsy showed that the diagnosis and location of the tumor had been correctly made. A tumor, nearly globular in form, measuring about an inch in its longest diameter, was found in the right lobe of the cerebellum, close to the peduncle. It was of rather soft consistence, and proved to be a gliosarcoma. It had compressed the convolutions of the cerebellum mainly upward and outward.

The chief interest in this case lies in endeavoring to trace the cause of the development of the tumor. A patient, the slave of excessive mental work for years, receives a sudden violent blow on the left side of the head, which causes bleeding from the left ear, tinnitus, and impaired hearing. Subsequently he begins to suffer from headaches, at first frontal but subsequently becoming general, and increasing in frequency and severity, until they become constant and are accompanied by nausea. Then follow vertigo on walking, tinnitus in the oppo site ear, and a sudden attack of homonymous diplopia, which is found to be due to paresis of both external recti muscles. Immediately succeeding the diplopia comes defective vision in both eyes, which is found to be due to papillitis with hæmorrhages. The vertigo increases, the patient apparently rotates toward the right side in walking, and the loss of vision and headaches become worse, till delirium sets in, ending in coma and death. The weak point in the relation of cause and effect, between the blow on the side of the head and the development of the cerebellar tumor, is that the traumatism occurred on the left side over the parietal and frontal bones, while the tumor was found on the right side.

TOXICOLOGY OF THE MALE FERN, WITH SPECIAL REFERENCE TO VISUAL DISTURBANCE.

BY DRS. K. KATAYAMA AND OKAMOTO.

We know the poisonous nature of the extract of male fern not from the cases of accidental poisoning alone; Puirll (*Diss. Berlin*, 1888), Paulson (*Archiv fr. experimentelle Pathologie u. Pharmakologie*, Bd. xxix, Heft 1 m. 2. S. 1) and Inoko (*Tokyo Iji Shinshi*, No. 689, 1891) have tried experiments on animals, and found that the drug causes irritation of the stomach and intestine, and that it also paralyzes the nerve centers.

We, too, have tried experiments on six dogs and four rabbits, and obtained almost similar results. As our object, however, is not to know the general toxic properties of the drug, but to ascertain its effect on the sense of vision, we shall here touch only briefly on the general symptoms of the poisoning in describing our experiments, and dwell principally on symptoms relating to the eye.

EXPERIMENTS ON ANIMALS.—The æthereal extract of male fern has a peculiar odor, and is not at all palatable; hence, dogs do not like it, and it is no easy matter to make them take it. In our experiments we made it into pills, tucked the pills into meat, and with all manner of persuasion and enticement, could barely succeed in administering the drug. It was a thing infeasible, as we have tried it, to have the dogs take each day a fixed quantity as we listed. It may be supposed that in the form of emulsion a fixed quantity of the drug can be introduced into the stomach by means of a tube. We have tried it, and found it impracticable to repeat the process several times upon the same dog.

DOG A. Age unknown. Body weight 13480 grains. Gave five decigrams to three grams (0.5-3.0) of the extract per day, kept on for seventy-three days, amounting in all to 47.2 grams. When about five decigrams (0.5) were being given, no effect on the dog could be noticed; but upon administering a dose much larger, vomiting and diarrhœa and exhaustion presented themselves. No injury came to the eye. After an interruption of ten days the experiment was resumed, and for sixty days two to eight decigrams (0.2-0.8) were given daily, coming up to 16.0 grams in the aggregate. For all this no derangement of stomach and intestine, nor an affection of the eye took place, nor any other disorder.

DOG B. Middle aged. Body weight, 4640 grams. Gave fifteen centigrams to seven decigrams (0.15-0.7) of the extract *per diem.* On the ninth day, when the total quantity exhibited reached up to 2.6 grams, tremor of the whole body appeared which lasted two or three days. During this period and before and after it no detriment overcome the eye. Hence forward in thirty days the sum of 7.75 grams was tried anew, and with intermissions the experiment was continued longer. But nothing came of it, except the refusal of food when a dose of the extract was large.

DOG C. A puppy, two months old. Body weight, 3640 grams. Gave on the first day four decigrams (0.4), and on the third seven decigrams (0.7), making in all 1.1 grams of the extract. On the fourth day already there appeared the signs of poisoning,—universal tremor, notable flexion of the spine, slow response to stimulation, and the curious trick of stretching the hind legs behind in the act of walking. To add to these, thirst was manifest. On the sixth day, the pupils were widely dilated, and they did not react; the axis of vision was turned upward; and the lids were widely open. In walking and running the creature bumped, from time to time, against the walls and chairs near by, and fell from the stairs. Although the threatening attitude of dealing a blow was assumed right in front of its eyes, the dog did not even as much as blink. And again, although a piece of meat was placed be-

fore him, the dog seemed not at all to know it. Only upon bringing it close to his nose did the dog seem to be, for the first time, aware of it—through the sense of smell. All this pointed to his being blind. Therefore, the further use of the extract was stopped.

A day or two after this, great debility followed, and the creature could well-nigh not drag himself. The condition of the eye staid pretty much the same. As the lids had remained open, slight conjunctivitis was set up, owing to the irritating action of the dust.

A week or so after the blindness had taken place, the pupils became somewhat contracted, vision returned a little, and the dog came to be able to get about. In another week he recovered his visual power to such an extent that he could spy out a piece of meat given to another dog and make for it. He continued to grow better, when one day he stole and ate a large quantity of meat preserved in alcohol that had been used in some other experiment. In consequence he suffered for a time from acute alcoholism, but recovered completely in two or three days under proper management. The dog still lives, healthy and strong. His vision is good; the pupils react to light perfectly, but they are somewhat larger than what they used to be before the experiment.

DOG D. A puppy, two months old, (of the same litter with Dog C). For the first three days five decigrams (0.5) of the extract of male fern were given daily. On the fourth day the whole body began to tremble, and action became slow to a degree. The opened eyelids, the dilated pupils and their want of reaction, the direction of visual axis, the loss of vision, etc., were almost same as in Dog C.

Two or three days after the loss of vision, the activity of gait returned somewhat, the pupils contracted a little, but sight improved not a particle. Besides, as the lids had remained open the dust caused irritation and excited slight inflammation of the conjunctiva, with increased secretion.

Nearly a week after the loss of vision, his actions became quite animated, yet the dog was none the less blind.

Some two weeks more, and the eye showed a tendency toward recovery; but like his erring brother, Dog C, this miscreant dog, too, practiced pilfering and had for that an attack of acute alcoholic poisoning. He could not get off so easily as the other dog, and eventually died of it; and we unfortunately could not follow out the entire course of the male fern poisoning.

DOG E. A puppy, about two months old. Body weight, 4120 grams. Having given twenty-five centigrams to five decigrams (0.25-0.5) daily, up to the twenty third day the sum of 5.15 grams had been given, of the extract of male fern. On the day mentioned, both pupils became dilated somewhat, but there seemed to be no interference with vision. In walking the hind legs were a little unsteady.

From now on, continuously and interruptedly 3.875 grams more had been given in thirty days, but nothing followed, the condition of the hind legs remaining the same.

DOF F. A puppy of an unknown age. Body weight, 1400 grams. Gave in eighteen days 3,875 grams of the extract, the daily dose being a hundred and twenty-five milligrams to five decigrams (0.125-0.5). During the last two or three days of the period the whole body, especially the head, shook involuntarily; an appearance of indisposition was visible; the hind legs were bereft of strength; and the act of walking was unsteady. Further, there were vomiting and purging; the strength of vision, too, seemed to have diminished a little, but the reaction of the pupils to light was retained. As it was feared that these symptoms might gradually become worse and the subject fall into collapse, the administration of the drug was discontinued for three days. Then at the rate of twenty-five centigrams to five decigrams (0.25-0.5) a day, 4.375 grams of the extract were given again in fourteen days. When a dose was rather large the dog refused to take food, and had vomiting and purging.

Beside these experiments on dogs, we gave four rabbits 2.0-5.0 grams of the extract in emulsion by means of a stom-

ach tube. The following are the chief symptoms that appeared:

An increased and shallow respiration; a loathing of food (no vomiting by nature); diarrhœa; an appearance of indisposition; inactivity; and but slow response to stimulation On giving a large quantity, the free motion of the legs was hindered; sometimes the legs presented a spasmodic motion. As to dilatation of the pupil and any disturbance of vision, although we have repeated the experiment several times, with one, two or five and six days between the aforementioned doses, we were not able to discover them.

From these our own experiments and those of others we conclude as follows:

1. The extract of male fern has a toxic property, and it acts principally on the digestive system and the nerve centers, producing such symptoms as vomiting, diarrhœa, colic, cephalalgia, difficulty of locomotion, dilatation of the pupil, impaired vision, hurried respiration, motor paralysis, depression, etc.

2. The main reason why we see more reports on poisoning by the extract of male fern lately than before, lies in the fact that in recent years the dose of the medicine has been increased considerably. As the quality of the medicine varies according to the degree of freshness of the preparation and the like, the fatal dose cannot well be determined. In general, however, when a large quantity is given there invariably appears an almost constant series of symptoms.

3. It is true that after the use of extract of male fern loss of vision sometimes appears, but it is not constant; only under a certain condition does it make its appearance.

4. Loss of vision is one of the symptoms of poisoning by the extract of male fern, and it is a matter of course that it may appear along with the other symptoms. However, it does not seem to be necessarily contingent upon the size of the dose and the mode of administration.

5. In looking over the various reports, we find that the patients who had failure of vision consequent to the use of the extract of male fern were generally persons of poor health.

The two dogs that became blind in our experiments were also young and weak. In general, amblyopia or amaurosis from poisoning (*e. g.*, by alcohol or by tobacco) is common in people of enfeebled constitution. From this we may rightly infer that the liability to amblyopia after the use of the medicine exists mostly in people of poor health. Not that persons of feeble health have all this liability alike, but that some among them must particularly be susceptible to the toxic action of the drug.

6. Such being the facts, therapeutists or practitioners of medicine would perhaps, do well to note the following points:

a. The extract of male fern is better to be prescribed in small doses.

b. The extract of male fern is readily absorbed when given mixed with oil. Even without oil, it is absorbed when it remains long in the alimentary canal.

c. After the extract of male fern, do not give castor oil. See to it that some other cathartic is given.

d When the extract of male fern is used, always look out for such symptoms as headache, amblyopia, etc.; and as soon as the slightest indications of these appear, stop the further use of the medicine.—*Sei-I-Kwai Medical Journal.*